

AIDS

THE SELENOENZYME SOLUTION

AIDS is a consequence of HIV infection which causes deficiencies of the enzyme glutathione peroxidase and its four components, yet this syndrome and viral activity can be reversed with dietary supplementation.

Part 2 of 2

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COROLLARY ONE: Deficiencies of Glutathione Peroxidase and its Components in HIV/AIDS

There is strong evidence to show that HIV-seropositive individuals are deficient in glutathione peroxidase. Gil and colleagues,⁵⁴ for example, compared levels of it in the blood of 85 HIV/AIDS patients with those in 40 healthy controls, confirming the presence of a significant ($p < 0.05$) reduction of the selenoenzyme in the infected group. Beyond this, Batterham and co-workers⁵⁵ showed that such depressed glutathione peroxidase levels in men with HIV/AIDS could be raised by supplementation with selenium and other antioxidants.

If Aumann and co-workers⁵⁶ are correct, then HIV/AIDS patients should also be very deficient in the four nutritional components that these researchers believe are required by the body to produce glutathione peroxidase—namely, selenium, cysteine, glutamine and tryptophan. There is certainly good evidence to prove that such individuals are selenium deficient.

Several studies have documented declining plasma selenium levels in patients with HIV/AIDS. Probably the most convincing of these was conducted by Baum and co-workers⁵⁷ in Florida. These researchers monitored 125 HIV-1-seropositive male and female drug users in Miami over a period of 3.5 years. This study collected data on CD4 T-cell count, antiretroviral treatment and plasma levels of vitamins A, E, B6 and B12 as well as selenium and zinc. A total of 21 of these patients died during the study. Only plasma selenium levels and CD4 T-cell counts could have been used to predict which of the 125 patients would die, with selenium levels being more accurate predictors than CD4 T-cell counts. The same research group also monitored 24 HIV-infected children over a five-year period, during which time half of them died of AIDS. As with adults, the lower their serum selenium levels, the faster that death occurred.

It also appears as if the selenium deficiency seen in HIV/AIDS patients, as expected, makes them more susceptible to Cocksackievirus infection. As a consequence, myocardial infarctions are quite common even in relatively young people who are HIV seropositive.⁵⁹ In addition, autopsies often reveal that AIDS patients^{60, 61} have been suffering from, and perhaps have died of, Keshan disease—an endemic heart disease normally limited to the populations of regions of extreme selenium deficiency.

HIV/AIDS patients also display low plasma levels of cysteine at every stage of infection.⁶² Since this amino acid is one of the body's major sources of sulphur, they are very deficient in it.⁶³ Interestingly, depressed cysteine is also characteristic of SIV-infected rhesus macaques.

Several researchers have documented glutamine deficiencies in HIV/AIDS patients.⁶⁵⁻⁶⁷ Shabert and colleagues, for example, discovered that much of the weight loss seen in individuals could be reversed by glutamine-antioxidant supplementation.

If HIV is producing glutathione peroxidase for its own purposes and if this selenoenzyme contains tryptophan, then HIV/AIDS patients should be deficient in this amino acid. This appears to be the case. Werner and co-workers,⁶⁸ for example, have shown that, in male patients with advanced HIV infection, tryptophan serum levels are less than half of those found in matched healthy controls. Since tryptophan is required for the biosynthesis of both serotonin and niacin, it is not surprising that their levels are also depressed in patients with HIV/AIDS.^{69, 70}

It is clear from the literature just cited that HIV/AIDS patients are indeed very deficient in glutathione peroxidase and in the four components of this selenoenzyme—namely, selenium, cysteine, glutamine and tryptophan. In short, the clinical and scientific evidence supports the truth of corollary one.

COROLLARY TWO: Effective Treatment for HIV/AIDS Should Involve Correcting Deficiencies of Glutathione Peroxidase and its Nutritional Precursors

There is a wealth of evidence that correcting one or more of the deficiencies of selenium, cysteine, glutamine and tryptophan, which are characteristic of HIV/AIDS, has significant health benefits. Selenium, for example, is a key immunological enhancement agent that has a strong impact on lymphocyte proliferation.

This relationship was confirmed by Peretz and co-workers,⁷¹ who monitored enhanced lymphocyte response in elderly subjects given a daily 100-microgram selenium supplement over a six-month clinical trial. This seems to be because selenium is essential for lymphocytes—as shown by Porter and colleagues,⁷² who demonstrated that plasma proteins carry selenium to lymphocytes which absorb it. Further, Wang and co-workers⁷³ have demonstrated that selenium enhances lectin-stimulated T-lymphocyte proliferation and is an important modulator for immune response. It is not surprising, therefore, that HIV/AIDS patients with depressed plasma selenium also show T-lymphocyte abnormalities.⁷⁴

There have been numerous clinical trials to explore the impact of cysteine supplementation (usually given as N-acetylcysteine) on HIV/AIDS symptoms. De Rosa and co-workers⁷⁶ at Stanford University, for example, have shown that the oral administration of N-acetylcysteine significantly replenished glutathione in HIV-infected individuals. This is very significant, since subsequent research has established that glutathione levels in HIV-positive patients is a predictor of survival rates.⁷⁷

As previously mentioned, cysteine is a significant source of sulphur and HIV/AIDS patients are very deficient in this element. A trial carried out in Germany by Breitzkreutz and colleagues⁷⁷ showed that N-acetylcysteine supplementation helped to correct this sulphur deficiency while simultaneously improving immunological functions in HIV/AIDS patients.

Glutamine is a major requirement of cells which are rapidly proliferating. As a result there is a significant requirement for it in the digestive tract, where it is essential for intestinal cell proliferation, intestinal fluid/electrolyte absorption and mitogenic response to growth factors. Since glutamine deficiency is so characteristic of HIV/AIDS, it is not surprising that patients typically suffer badly from digestive malfunction and diarrhoea. It has been demonstrated by Noyer and co-workers,⁷⁸ at the Albert Einstein College of Medicine, that glutamine therapy improves intestinal permeability in AIDS patients, although the amount required to enhance intestinal absorption may be as much as 20 grams per day.

Glutamine is also essential for muscle building; in HIV/AIDS patients, deficiencies of it seem linked to loss of body cell mass. Shabert and his colleagues⁷⁹ have demonstrated that glutamine and antioxidant supplements can reverse the weight loss typically seen in such patients, while Kohler and co-workers⁸⁰ also have

shown that glycyl-glutamine improves lymphocyte proliferation in AIDS patients.

I am not aware of any clinical trials conducted to test the impact of tryptophan supplementation on HIV/AIDS. However, it is interesting to note that antiretroviral drug therapy, designed to prevent HIV-1 replication, slows the rate of tryptophan loss seen in seropositive individuals.⁸¹ Similarly, plasma tryptophan levels can be increased in HIV-infected patients by nicotinamide supplements.⁸² This is perhaps not surprising, given the close chemical association between this nutrient and the tryptophan derivative, niacin.

Simply put, there is a great deal of evidence that HIV/AIDS patients are typically deficient in glutathione peroxidase and its precursors—selenium, cysteine, glutamine and tryptophan. Beyond this, it is clear from clinical trials that survival rates and patients' symptoms are improved by supplementation with such nutrients.

Indeed, one might go so far as to say it would be medical malpractice *not* to give these nutrients to those who are HIV seropositive.

COROLLARY THREE: Reversing Deficiencies of the Precursors of Glutathione Peroxidase Should Reverse the Symptoms of HIV/AIDS

The hypothesis presented here suggests that HIV/AIDS is a disease that is caused by the combined deficiencies of glutathione peroxidase and its precursors. If this is correct, then the symptoms normally associated with a deficiency of each one of these substances ought to occur in AIDS patients. There is a wealth of evidence that suggests this is the case.

Baum and co-workers⁸³ have shown that adults and children dying of AIDS display both depressed CD4 T-

lymphocyte counts and very depleted plasma selenium stores. This seems to be part of a positive feedback system, since one of the most significant symptoms of selenium deficiency is a reduction of CD4 T-lymphocytes, which occurs because this trace element is needed for their production. A lowering of CD4 T-lymphocyte levels causes a drop in the efficiency of the immune system, encouraging infection by other pathogens and resulting in a further decline in selenium. I have termed this positive feedback system the *selenium CD4 T-cell tailspin*.⁸⁴

HIV/AIDS patients also often display a hypothyroid or low T3 (tri-iodothyronine) syndrome.⁸⁵ This seems to occur because selenium deficiency causes a reduction in deiodinase, the enzyme required to convert T4 (thyroxine) to T3. It has been further suggested that such a selenium deficiency abnormality of the thyroid may be a significant factor in the AIDS wasting process.⁸⁶

Selenium deficiency has been linked to depression in the general population.^{87,88} It is not surprising, therefore, that this is also a characteristic of people with HIV/AIDS.

It would appear, therefore, that at least three of the major symptoms of HIV/AIDS—namely, depressed CD4 T-lymphocyte count, lowered tri-iodothyronine production and depression—can

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be explained, at least in part, by the inadequate selenium levels seen in such patients.

In 1981, Bunk and Combs⁸⁹ described an experiment demonstrating that, in chickens, selenium deficiency impaired the conversion of the S-amino acid methionine into cysteine. It is highly likely that this is true for humans. If it is, then, by encoding for the selenoenzyme glutathione peroxidase, HIV-1 causes a deficiency of cysteine in infected individuals in two distinct ways. Firstly, the virus removes cysteine directly from the body as it replicates. Secondly, it creates a selenium deficiency which impairs the conversion of methionine to cysteine, so reducing the availability of the latter. Simply put, HIV-1 both increases the demand for and reduces the supply of cysteine in patients who are HIV-1 positive. Cysteine deficiency, in and of itself, has been shown to be associated with depressed glutathione, poor wound and skin healing, psoriasis, abnormal immune function and greater susceptibility to secondary infections and cancers.⁹⁰ All these characteristics of cysteine deficiency are seen in HIV/AIDS patients.

Glutamine is a major nutrient required by rapidly proliferating cells and is of particular significance in the digestive tract.

Deficiencies cause abnormal intestine permeability and digestive malfunction, often associated with diarrhoea.⁹¹ Glutamine is also a favourite with body-builders, who use it in large quantities to promote muscle growth. It is not surprising that muscle protein wasting, therefore, is a symptom of glutamine inadequacy. Both diarrhoea and muscle wasting are characteristics of HIV/AIDS.⁹²

Tryptophan deficiencies, in and of themselves, have led to major health problems in the past. Probably the worst of these was pellagra, which developed in children eating diets high in corn. Maize is very deficient in

tryptophan and so such children quickly developed pellagra, which is thought to be due to a co-deficiency of both tryptophan and its metabolite, niacin.⁹³ As a consequence of these two deficiencies, such individuals could not produce adequate nicotinamide adenine dinucleotide and so developed pellagra. The symptoms of this disease were known as "the four Ds"—namely, dermatitis, diarrhoea, dementia and, ultimately, if not treated effectively, death.⁹⁴ AIDS patients commonly experience all such symptoms and also display inadequate levels of nicotinamide adenine dinucleotide. This can be reversed, at least *in vitro*, by the administration of nicotinamide.⁹⁵

It would appear, therefore, that corollary three is correct and that the great majority of the symptoms of HIV/AIDS (with the exception of those caused by opportunistic pathogens) are a combination of symptoms seen in individuals who are extremely deficient in glutathione peroxidase or in one or more of its precursors.

COROLLARY FOUR: HIV-1 Seropositive Individuals Who Eat a Diet Elevated in Selenium, Cysteine, Glutamine and Tryptophan Should Never Develop AIDS

Obviously, the easiest way to test the truth or otherwise of this fourth corollary would be to arrange for a double-blind, placebo-controlled pilot study in which half the HIV/AIDS patients are

given injections of glutathione peroxidase and supplements of selenium, cysteine, glutamine and tryptophan.

Unfortunately, geographers are not expected to develop new disease-related hypotheses that have the potential for undermining genetic, biochemical and clinical authority. As a result, I have been attempting to gain support for testing this concept for more than two years. Given the enormous power of the pharmaceutical industry and its lack of interest in the discovery of a cheap and simple treatment for HIV/AIDS, it has not been an easy row to hoe. To date, all I can point to are two AIDS patients who quickly reversed their major symptoms when attempting to follow my suggested regime.⁹⁶ Beyond this, there are research teams in South Africa, Tanzania, Botswana and Morocco who have contacted me to express a willingness to conduct such trials, should funding ever become available.

CONCLUSIONS

Death from AIDS is a consequence of four nutritional deficiencies. Fortunately, HIV infection does not need to be a death sentence because such deficiencies are cheap and easy to reverse. And while the four nutrients won't eradicate HIV, they

activate the virus's own "warning system", preventing its replication.

The genetic code of HIV includes a homologue for the essential human selenoenzyme glutathione peroxidase. Paradoxically, this viral requirement for selenium generally appears to restrict infection to individuals who, because of a diet deficient in selenium or because of prior infection by other selenium-encoding pathogens, are deficient in this trace element.

Unfortunately, the human population is becoming ever more susceptible to infection by HIV-1 (and HIV-2 to a lesser extent) as well as other selenoenzyme-encoding viruses

because of acid rain, which reduces the bioavailability of selenium.

To be replicated, HIV must compete with its host for glutathione peroxidase and its four constituent nutrients—selenium, cysteine, glutamine and tryptophan. As a consequence, replication of the virus gradually depletes seropositive individuals of these substances. AIDS is the end product of these nutritional declines, and most of its symptoms are caused by them. As a consequence, it is likely that AIDS can be easily reversed by correcting such deficiencies.

To illustrate, glutathione peroxidase is one of the body's most significant antioxidants. A lack of this selenoenzyme therefore accelerates free radical damage and oxidative stress. Beyond this, having inadequate selenium and cysteine undermines the immune system in a process that is accelerated by other infectious pathogens. A deficiency of glutamine encourages muscle wasting and digestive malfunction, while a lack of tryptophan and the compounds it biosynthesises (such as niacin and serotonin) results in dermatitis, diarrhoea and various neurologic and psychiatric symptoms including dementia. Supplementation with the appropriate nutrients naturally reverses these symptoms.

It is ironic, but not really surprising, that our continuous destruction of the global ecosystem is promoting the spread of viral infections (and various chronic degenerative diseases) that threaten humanity's domination of the planet.

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POSTSCRIPT (as at early January 2004)

Since I submitted this article for publication, I have learned of a small AIDS trial that is taking place in Botswana.⁹⁷ The trial is funded by a Canadian vitamin company and is using the nutrient regimen suggested in my book. Here is a quotation from the initial email report that I received in late September:

"I picked two candidates personally who have fully blown AIDS with relevant symptoms like diarrhoea, skin rash, loss of weight and a lack of appetite. One of these candidates has a severe complication of syphilis which has slowed his recovery somewhat, but still, within two weeks of trials, his skin rash, diarrhoea and fatigue have all but disappeared. The lady candidate gained 3 kg in two weeks and now eats 'like a horse'. She resumed work last Tuesday after several weeks of absence. I am gaining confidence in this treatment by the day and I hope the same would apply to the remainder of the trial candidates..."

"A lady who started the regimen three weeks back has just tested negative for HIV, and her CD4 count has shot up from 500 to 700!" (It's unknown if this is the same lady who ate "like a horse"!)

In the meantime, I have set up a small company, HD Foster Research Inc., which is having the nutrients made up into a product called HELP. We are giving this away to doctors who treat AIDS patients. The first taker is a physician in South Africa, and I have mailed him enough treatment for 10 patients. The idea is to find medical supporters who can vouch that the treatment works. Beyond this, the small Canadian company that is using my treatment in Botswana (anecdotal evidence suggests a 99% success rate in reversing AIDS) has spread its activities into Zambia.

We have decided to produce a video in which I describe my theory of HIV/AIDS, and which also shows patients recovering. We are looking for financial and other assistance to do this. The idea is to give this away to TV stations in Africa and elsewhere.

Recently I checked the progress of the two Victoria, BC, patients mentioned in my book, who were dying of AIDS in 2001. They are now both in good health and are back at work.

I have also had two more HIV/AIDS papers published in Chinese in the proceedings of two different medical conferences held in Shanghai in November 2003. Two additional papers have been accepted for publication in Chinese medical journals. On 17 March I am scheduled to give a lecture on AIDS at the Centennial AGM of the Association of American Geographers in Philadelphia.

Things are moving along. Hopefully, the world will soon know that the treatment does indeed work.

Editor's Note:

Readers wanting more detailed information about the HIV/AIDS environmental connection are directed to the website <http://www.hdfoster.com>, where they can download a free copy of Harold Foster's book, *What Really Causes AIDS*.

About the Author:

Harold D. Foster, PhD, was born and educated in England. He specialised in geology and geography, earning a BSc in 1964 from University College London and a PhD in 1968 from London University. He is a Canadian by choice, and has been a faculty member in the Department of Geography, University of Victoria, British Columbia, Canada, since 1967.

A tenured professor, Dr Foster has authored or edited some 235 publications, the majority of which focus on reducing disaster losses or identifying the causes of chronic disease or longevity. He has published hypotheses on the origins of numerous diseases including myocardial infarction, AIDS, cancer, diabetes, schizophrenia, multiple sclerosis (MS), amyotrophic lateral sclerosis (ALS), Alzheimer's and Parkinson's diseases, and stroke.

His numerous books include: *Disaster Planning: The Preservation of Life and Property* (Springer Verlag, New York, 1980); *Reducing Cancer Mortality: A Geographical Perspective* (Western Geographical Press, Victoria, 1986); *The Ozymandias Principles: Thirty-one Strategies for Surviving Change* (Southdowne Press, Victoria, 1997); and *What Really Causes AIDS* (Trafford Publishing, Victoria, 2002; see review in NEXUS 10/05). His new book, *What Really Causes Schizophrenia* (Trafford, 2003), is reviewed in this issue of NEXUS.

Harold Foster is a member of the Explorers Club as well as several academic organisations including The New York Academy of Sciences, The Royal Geographical Society and The Royal Society of Literature. He is also the editor of both the International and Canadian *Western Geographical Series* and is a member of the boards of the *Journal of Orthomolecular Medicine* and the International Schizophrenia Foundation.

He has been a consultant to numerous organisations, including the United Nations and NATO, and to the governments of Canada, Ontario and British Columbia. He is also a member of the Science Advisory Panel for the Healthy Water Association.

Every day, Dr Foster makes a point of taking at least the recommended daily allowance of the known essential nutrients. He is also currently pursuing offers for his suggested nutrient mixture to be produced for use in clinical trials with AIDS patients. For a more detailed résumé, visit the website <http://www.hdfoster.com>.

Endnotes

54. Gil, L. and others, "Contribution to characterization of oxidative stress in HIV/AIDS patients", *Pharmacol Res* 2003; 47(3):217-224.
55. Batterham, M. and others, "A preliminary open label dose comparison using an antioxidant regimen to determine the effect on viral load and oxidative stress in men with HIV/AIDS", *Eur J Clin Nutr* 2001; 55(2):107-114.
56. Aumann, K.D. and others, "Glutathione peroxidase revisited – simulation of the catalytic cycle by computer-assisted molecular modeling", *Biomed Environ Sci* 1997; 10(2-3):136-155.
57. Baum, M.K. and others, "High risk of HIV-related mortality is associated with selenium deficiency", *J Acquir Immune Defic Syndr Hum Retrovirol* 1997; 15(5):370-374.
58. Campa, A. and others, "Mortality risk in selenium deficient HIV-positive children", *J Acquir Immune Defic Syndr Hum Retrovirol*

1999; 20(5):508-513.

59. Law, M. and others, "Modelling the 3-year risk of myocardial infarction among participants in the Data Collection on Adverse Events of Anti-HIV Drug (DAD) study", *HIV Med* 2003; 4(1):1-10.
60. Dworkin, B.M., "Selenium deficiency in HIV infection and the acquired immunodeficiency syndrome (AIDS)", *Chem Biol Interact* 1994; 91(2-3):181-186.
61. Dworkin, B.M. and others, "Reduced cardiac selenium content in the acquired immunodeficiency syndrome", *J Parenter Enteral Nutr (JPEN)* 1989; 13(6):644-647.
62. Droge, W. and others, "Functions of glutathione and glutathione disulfide in immunology and immunopathology", *FASEB J* 1994; 8:1131-1138.
63. Breitkreutz, R. and others, "Improvement of immune functions in HIV infection by sulfur supplementation: two randomized trials", *J Mol Med* 2000; 78(1):55-62.

64. Droge, W. and others, "HIV-induced cysteine deficiency and T-cell dysfunction – a rationale for treatment with N-acetylcysteine", *Immunol Today* 1992; 13(6):211-214.
65. Shabert, J.K. and others, "Glutamine-antioxidant supplementation increases body cell mass in AIDS patients with weight loss: a randomized double-blind controlled trial", *Nutrition* 1999; 15(11/12):860-864.
66. Noyer, C.M. and others, "A double-blind placebo-controlled pilot study of glutamine therapy for abnormal intestinal permeability in patients with AIDS", *Am J Gastroenterol* 1998; 93(6):972-975.
67. Kohler, H. and others, "Glycyl-glutamine improves *in vitro* lymphocyte proliferation in AIDS patients", *Eur J Med Res* 2000; 5(6):263-267.
68. Werner, E.R. and others, "Tryptophan degradation in patients infected by human immunodeficiency virus", *Biol Chem Hoppe*

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Seyler 1988; 369(5):337-340.

69. Murray, M.F. "Niacin as a potential AIDS preventative factor", *Med Hypotheses* 1999; 53(5):375-379.

70. Sidibe, S. and others, "Effects of serotonin and melanin on *in vitro* HIV-1 infection", *J Biol Regul Homeost Agents* 1996; 10(1):19-24.

71. Peretz, A. and others, "Lymphocyte response is enhanced by supplementation of elderly subjects with selenium-enriched yeast", *Am J Clin Nutr* 1991; 53(5):1323-1328.

72. Porter, E.K. and others, "Uptake of selenium-75 by human lymphocytes *in vitro*", *J Nutr* 1979; 109(11):1901-1908.

73. Wang, R.D. and others, "Investigation of the effect of selenium on T-lymphocyte proliferation and its mechanisms", *J Tongji Med Univ* 1992; 12(1):33-38.

74. Baum, M.K. and others, "High risk of HIV-related mortality is associated with selenium deficiency", *J Acquir Immune Defic Syndr Hum Retrovirol* 1997; 15(5):370-374.

75. De Rosa, S.C. and others, "N-acetylcysteine replenishes glutathione in HIV infection", *Eur J Clin Invest* 2000; 30(10):915-929.

76. James, J.S., "NAC: First Controlled Trial, Positive Results", *AIDS Treatment News* 1996; 250:1-3, posted at <http://www.aids.org/immunet/atn.nsf/page/ZQX25002.html>.

77. Breitkreutz, R., "Improvement of immune

functions in HIV infection by sulfur supplementation: two randomized trials", *J Mol Med* 2000; 78(1):55-62.

78. Noyer, C.M. and others, "A double-blind placebo-controlled pilot study of glutamine therapy for abnormal intestinal permeability in patients with AIDS", *Am J Gastroenterol* 1998; 93(6):972-975.

79. Shabert, J.K. and others, "Glutamine-antioxidant supplementation increases body cell mass in AIDS patients with weight loss: a randomized double-blind controlled trial", *Nutrition* 1999; 15(11/12):860-864.

80. Kohler, H. and others, op. cit.

81. Zangerle, R. and others, "Effective antiretroviral therapy reduces degradation of tryptophan in patients with HIV-1 infection", *Clin Immunol* 2002; 104(3):242-247.

82. Murray, M.F. and others, "Increased plasma tryptophan in HIV-infected patients treated with pharmacologic doses of nicotinamide", *Nutrition* 2001; 17(7-8):654-656.

83. Baum, M.K., op. cit.

84. Foster, H.D., "AIDS and the 'selenium-CDR T cell tailspin': The geography of a pandemic", *Townsend Letter for Doctors and Patients* 2000; 209:94-99.

85. Bourdoux, P.P. and others, "Biochemical thyroid profile in patients infected with the human immunodeficiency virus", *Thyroid* 1991; 1:149.

86. Geelhoed-Duijvestijn, P.H. and others, "Effect of administration of growth hormone on

plasma and intracellular levels of thyroxine and tri-iodothyronine in thyroidectomized thyroxine-treated rats", *J Endocrinol* 1992; 133:45-49.

87. Hawkes, W.C. and others, "Effect of dietary selenium on mood in healthy men living in a metabolic research unit", *Biol Psychiatry* 1996; 39:121-128.

88. Finley, J.W. and others, "Adequacy or deprivation of dietary selenium in healthy men: clinical and psychological findings", *J Trace Elem Exp Med* 1998; 11:11-27.

89. Bunk, M.J. and others, "Evidence for an impairment in conversion of methionine to cysteine in the Se-deficient chicken", *Proc Soc Ex Biol Med* 1981; 167:87-93.

90. Braverman, E.R. (with C.C. Pfeiffer), *The Healing Nutrients Within: Facts, Findings and New Research on Amino Acids*, Keats Publishing, New Canaan, 1987.

91. Rhoads, M., "Glutamine signalling in intestinal cells", *J Parenter Enteral Nutr* 1999; 23(5 Suppl):S38-40.

92. Ward, D.E., *The AmFAR AIDS Handbook: the Complete Guide to Understanding HIV and AIDS*, W.W. Norton, New York, 1999.

93. Braverman, E.R., op. cit.

94. *ibid.*

95. Murray, M.F. and others, "HIV infection decreases intracellular nicotinamide adenine dinucleotide (NAD)", *Biochem Biophys Res Commun* 1995; 212(1):126-131.

96. Foster, H.D., 2000, op. cit.

97. Email to author, September 25, 2003.